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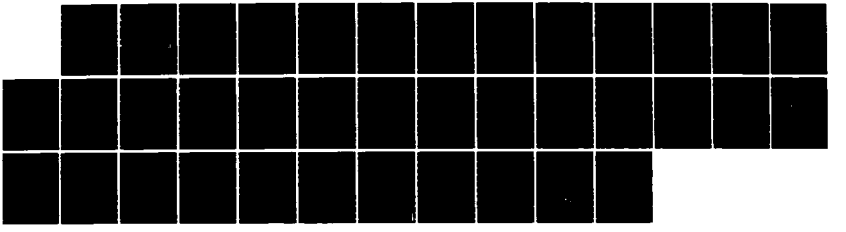

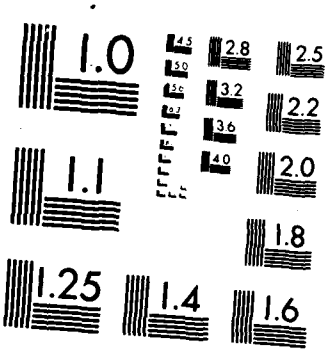

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a linear relation was observed between PO2 and PCO2 in both arterial and alveolar samples. A linear relation was also observed for PO2 and Pb, and PCO2 and Pb for both arterial and alveolar samples. Summit mean alveolar PO2=29.8± 3.4 and PCO2=12.4±1.9. Summit mean arterial PO2=29.3±2.4 and PCO2=11.7±1.5. This study describes for the first time the relation between PO2, PCO2, and Pb from sea level to extreme hypoxia (PB=240 torr) in a large group of subjects with both arterial and alveolar gases.

OPERATION EVEREST II: ALVEOLAR AND ARTERIAL BLOOD GASES AT EXTREME ALTITUDE

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Abstract

The relationship between alveolar PO2 and PCO2 at altitudes up to the summit of Mt. Everest (barometric pressure (Pb)=240 torr) has been investigated in a small number of individuals during mountaineering expeditions and one hypobaric chamber study. No previous study has measured these values on the same subjects throughout an entire ascent and confirmed their results with arterial blood gases. Alveolar gases were measured daily on eight healthy male subjects in a decompression chamber from sea level to a simulated summit of Mt. Everest (Pb=240torr). Arterial blood gases were measured on eight occasions. Below 575 torr, a linear relation was observed between PO2 and PCO2 in both arterial and alveolar samples. A linear relation was also observed for PO2 and Pb, and PCO2 and Pb for both arterial and alveolar samples. Summit mean alveolar PO2=29.8±3.4 and PCO2=12.4±1.9. Summit mean arterial PO2=29.3±2.4 and PCO2=11.7±1.5. This study describes for the first time the relation between PO2,PCO2,and Pb from sea level to extreme hypoxia (Pb=240 torr) in a large group of subjects with both arterial and alveolar gases.

Index Terms- Hypoxia, Hyperventilation, Decompression Chamber Oxygen, High Altitude

OPERATION EVEREST II: ALVEOLAR AND ARTERIAL BLOOD GASES AT EXTREME ALTITUDE

Increased pulmonary ventilation is a crucial adaptation to chronic hypoxemia no matter whether the hypoxemia results from disease or from residence at high altitude. The limits of this adaptive process are not clearly known. For example, persons with emphysema and chronic bronchitis may suffer from severe hypoxemia, but the ability to increase ventilation may be limited by mechanical factors, or in the case of chronic bronchitis, by impaired chemosensitivity. Healthy persons who ascend to the highest possible terrestrial elevation provide a unique approach to the problem, but measurements in such persons are few, and those that have been made are not in close agreement. In a previous chamber study of simulated altitude, 2 of 4 subjects tolerated decompression over 4 weeks to a barometric pressure (Pb) of 235 torr (14). In that study alveolar carbon dioxide tension, an index of ventilatory adaptation fell to 13 torr. In 1957 Pugh measured alveolar gases during a mountain expedition at Pb 347, 337, and 308 torr. He found an alveolar PO2 of 34 and PCO2 of 17 torr at Pb 308 torr. During the Silver Hut Expedition in 1962 Gill measured at Pb 288 torr an alveolar PO2 32.8 and PCO2 14.3 torr. The next report of alveolar gases at extreme altitude was from the 1981 American Medical Research Expedition to Mt. Everest (AMREE), where alveolar PO2 of 37.6 and PCO2 of 7.5 torr were measured in one subject who had climbed to the summit (8848 m, 253 torr)(19). Calculated arterial pH (7.75

units) from this alveolar sample indicated severe alkalosis. Needed were repeated measurements of the respiratory gases in blood and alveolar air from the same individuals ascending from sea level to the near limit of human tolerance to hypoxia.

An approach to these problems was made possible by Operation Everest II, where eight healthy young men volunteered to be confined for 6 weeks of decompression in a hypobaric chamber to the simulated summit of Mt. Everest. The utilization of a chamber provided a safe, controlled environment where careful and repeated measurements could be made. Apart from hypoxia, the chamber experiment was intended to eliminate the rigorous conditions on the mountain, while providing an opportunity for comparison of ventilatory adaptation with previously published data. It was expected that the results would provide unique and important information on respiratory adaptation to severe chronic hypoxia.

METHODS

The studies were conducted on 8 healthy males at sea level during a 40 day gradual decompression in a hypobaric chamber to the equivalent summit of Mt. Everest (PIO_2=43 torr) and then upon return to sea level. One subject was removed from the study at Pb 380 and another at Pb 280 torr because of transient hypoxic cerebral episodes. Alveolar and end tidal gases were sampled twice nearly every day throughout the ascent and for 72 hours after return to sea level. Arterial blood gases were sampled on eight occasions. Arterial blood gases at Pb 760,482,428, and

349 torr were sampled within several hours from the alveolar samples reported on that day. Arterial blood gases at Pb 308,282,252, and 240 torr were sampled immediately before the alveolar samples reported on that day.

Immediately after rising each morning alveolar and end tidal gas sampling on each subject was performed. The subjects sat in a comfortable position on a chair and breathed through an 8 by 3 centimeter cylindrical disposable mouthpiece with a mass spectrometer sampling capillary tube inserted into the mouth piece 4 centimeters from the subjects mouth. Noseclips were worn by the subjects to insure all expired gases were measured in the mouthpiece. The mass spectrometer was calibrated with standardized gases before each set of samples.

After several minutes of quiet breathing, sampling of expired gases (dry) was begun by a Perkin Elmer 1100 B mass spectrometer (Perkin Elmer, Burlington MA) and recorded on a DEC MINC II (Digital Electronics Corporation , Maynard MA) computer every 0.1 seconds for 30 seconds. After several tidal breaths during the sampling period, when ventilation appeared stable the subject was instructed to expire at end inspiration as completely and rapidly as possible following the method of Haldane and Priestly (11).

The recorded values of CO2 and O2 percent were then plotted by the computer against time. The resultant curve was examined to insure that tidal breathing was stable and expiration had been as complete as possible (reflected by a plateau of CO2 and O2 values). The end tidal values were defined as the lowest value

of O2 and highest values of CO2 averaged from at least two tidal breaths. The alveolar values were defined as the lowest value of O2 and the highest value of CO2 during the forced expiration. The mass spectrometer measured the gases as a percent of a dry gas. The percent values obtained were then multiplied by < barometric pressure - water vapor pressure (47 torr)> to achieve the partial pressure of CO2 and O2.

Arterial blood gases were collected on eight occasions, via an indwelling brachial or radial arterial catheter. After withdrawing 5 cc of blood to clear the catheter, arterial samples were collected in heparinized glass syringes and analyzed in duplicate immediately using a Radiometer ABL 3 (Woburn, MA) automated blood gas analyzer. Before each set of arterial samples, the ABL 3 was calibrated within expected ranges for arterial PCO2 and PO2 with both dry gases and tonomotored blood.

Inspired oxygen pressure in the trachea, when saturated with water vapor at body temperature is 43 mm of mercury-calculated on the basis of 253 torr barometric pressure on the summit of Mt. Everest (19). This assumes an oxygen concentration of 20.93%. In the present study at the extreme altitude inspired oxygen increased to 22.00% due to the oxygen exhaled by 6 scientists who were breathing oxygen-enriched air. This necessitated decompressing the chamber to the barometric pressure of 240 torr in order to ensure a PIO2 of 43 mm of mercury.

The 6 subjects who tolerated the lowest barometric pressure were initially decompressed to the summit on day 34 and then returned to the summit individually and in groups on days 35-40. Values reported from days 35-40 represent a

compilation of results from individual ascents to the summit for testing.

RESULTS

With decompression of the chamber over 40 days from sea level to a barometric pressure of 240 torr, the alveolar,end tidal and arterial oxygen and carbon dioxide tensions decreased (Tables 1 and 3) Table 1 shows the mean values of expired and arterial gases on each day samples were taken. Altitude is reported in barometric pressure. Values represent the means of eight subjects until 21 Oct., seven subjects until 7 Nov., and six subjects from 8 Nov. on. Six subjects were taken to the summit on 8 Nov. From Nov. 9-14, subjects were taken to the summit individually and in small groups for various studies. Values reported during these dates represent the results for the six subjects during their individual testing. Summit mean alveolar PO2 = 29.8 $\pm$ 3.4 and PCO2 = 12.41 $\pm$ 1.9 torr. Summit mean arterial PO2 = 29.33 $\pm$ 2.37 and PCO2 = 11.68 $\pm$ 1.5 torr. Table 3 shows the individual results of arterial blood gases from sea level to the summit. Arterial pH reached a maximum of 7.56 $\pm$.02 units.
The mean PaCO2 of 33.17 at sea level probably reflects hyperventilation due to anxiety as this sample was taken during the first invasive procedure on each subject.

Figures 1A and 1B show close correlation between individual arterial and alveolar samples. On close examination of the data it becomes apparent that the alveolar PO2 values are several torr lower than the arterial PO2. Figures 2A and 2B show close correlation between mean values of alveolar and end tidal samples

as would be expected.

Figure 3A shows the relationship between PCO2 and PO2 for both alveolar and arterial samples from sea level to a barometric pressure of 240 torr. The subjects were decompressed from sea level to a barometric pressure of 575 torr during the first twenty four hours of decompression. Although the mean alveolar PO2 dropped from 104.3 torr ($\pm$9.9) to 63.2 torr ($\pm$9.0), the PCO2 did not change (38.1 $\pm$ 4.3 and 38.3 $\pm$3.4). Below a barometric pressure of 575 torr, decreases in barometric pressure were less drastic to allow for acclimatization and to simulate an actual mountain ascent. Figure 3B shows the PCO2 PO2 relation below Pb=575 torr, which was linear.

The relationship between PO2 and barometric pressure and PCO2 and barometric pressure for both arterial and alveolar samples below a barometric pressure of 575 torr is shown in Figure 4. Linear relationships are observed between PO2 and Pb, and PCO2 and Pb for both arterial and alveolar samples.

From Table 1 and Figure 5 it is evident that alveolar samples taken at the same barometric pressure at different times can be compared. There were no statistically significant changes in alveolar PO2 and PCO2 over time at the same barometric pressure.

Table 2A and Figure 6 include the results of alveolar gas samples upon returning to sea level for the six subjects who successfully made the summit. At 72 hours, the subjects were still hyperventilating. The subject who was removed at 25,000 feet had sea level alveolar gases and arterial gases up to 10 days after return to sea level. Results shown in Table 2A and Figure 6 showed significant hyperventilation in this subject for 9 days

after return sea level.

Figure 7 compares the mean alveolar gases of the seven subjects who were decompressed to 282 torr with the subject who had to be removed at 380 torr. Despite being a competitive endurance athlete he did not show as great a ventilatory response to hypobaric hypoxia as the other subjects.

DISCUSSION

This study describes the resting ventilatory response to progressive hypobaric hypoxia at rest at increasing altitudes. It demonstrates a linear relationship between PO2 and PCO2, between PCO2 and barometric pressure and PO2 and barometric pressure below 575 torr. Although ventilation increases as the partial pressure of ambient oxygen decreases, it is not sufficient to prevent PO2 from decreasing, eventually to a level where life cannot be sustained.

Hypobaric chamber measurements allow for maximum data accuracy and reliability unlike measurements made on the mountain. Frequent calibration and standardization of measuring instruments were performed. Before measuring each set of alveolar samples the mass spectrometer was calibrated with gases of known concentration. Below Pb 288 torr, before each set of alveolar samples, bag samples of three different subject's alveolar air were measured on the mass spectrometer, a second mass spectrometer and on the ABL blood gas machine and showed nearly identical results. Before each set of arterial blood samples the ABL 3 blood gas machine was calibrated with tonometered blood that came from gas measured on the Schollander

apparatus. Arterial blood gas analysis was done promptly. Alveolar gases appear reliable in that they closely agree with the arterial blood gases, (especially at the lower barometric pressures when the arterial samples were taken in juxtaposition with the alveolar samples) although for 5 out of 8 measurements the alveolar PO2 is several torr lower than the arterial PO2. During steady state conditions the opposite would be expected. Although the arterial samples were taken under steady state conditions, the forced expiratory maneuver performed to collect alveolar samples preclude a steady state. During the forced expiratory maneuver, it is likely that O2 was constantly removed from the alveolus. Also alveolar air composition is not uniform throughout the lung and the gas collected from the forced expiratory maneuver is effected by regional variations in alveolar air composition. Thus the A-a gradient calculated from the alveolar gas equation is not likely to be obtained from near simultaneous arterial and alveolar samples. This has been shown previously (14). From Figure 8 it is apparent that at the moderate altitudes the data closely agrees with previous studies. All measurements were made on at least six individuals even at the extreme altitudes.

Figure 8 compares the results of the data presented in this paper with previous studies of alveolar gases at altitude. In general the data presented in this paper agrees with that of previous studies. It is apparent that in the first few days of this study the subjects were not acclimatized accounting for the lack of increased ventilation to hypoxia, described by Fitzgerald

who studied permanent residents at moderate altitude (8). The present study was limited by the constraints of having subjects confined in a decompression chamber and thus the initial ascent was made rapidly, followed by a much more gradual ascent profile allowing for acclimatization. One of the difficulties with most previous studies is they provide data on only a few points on the figure shown. Rahn and Otis (17) attempted to group 12 studies (16 data points) together to describe the PO2 PCO2 relation from sea level to 310 torr. The limitation with describing the PO2 PCO2 relationship with compiling different studies is that they are non homogeneous groups , with different durations at altitude. At the more extreme hypoxic ranges data is from only one or two individuals, and thus it is difficult to know how representative they may be. Pugh in 1957 provided data at Pb 347,337,and 308 torr and Gill in 1962 provided data at Pb 344,300 and 288 torr and in general agree with the findings of the present study (9,16) (Tables 1,4). The data from West in 1981 on one subject on the summit of Mt. Everest differed significant (Tables 1,4). He reported a PAO2 of 37.6 and PCO2 of 7.5 mm mercury (mean of four samples) The subject reported great difficulty in delivering the alveolar samples, which may explain the range for PACO2 of 5.1-9.0 for the four samples. The arterial pH data reveal the subjects to be less alkalemic than previously theoretically calculated (19). The pH values show a modest rise and suggest that over time despite the fall in PCO2 there was a considerable compensatory metabolic acidosis with bicarbonate excretion and appeared to stabilize at a pH of approximately 7.55. This is considerably

less alkalemic than the value of 7.75 reported by West et al. of an estimated value of pH for the summit of Mt. Everest (19). The discrepancy may be related to inadequate acclimatization in our subjects. However, it is not likely that the lack of metabolic compensation reported by West et al is consistent with acclimatization. Another possibility is that the assumptions used in their calculated pH would not be correct for these experimental conditions.

This study showed continued hyperventilation at 72 hours after return to sea level in the six successful "summitees". Of particular note, is the persistent hyperventilation 9 days later in the one subject removed at barometric pressure of 280 Torr. Hyperventilation upon return to sea level for this period of time implies an altered ventilatory drive that persists. Arterial blood gas analysis on return to sea level showed continued primary respiratory alkalosis as opposed to compensatory respiratory alkalosis from the metabolic acidemia developed at altitude.

It is interesting to note the different ventilatory response from the subject who collapsed early in the study. This subject demonstrated a lesser ventilatory response to hypoxia than the other subjects up to the day he collapsed (Figure 7).

What is the significance of the data presented? The alveolar PO2 PCO2 relation from sea level to extreme hypoxia was determined and in general confirmed previous data but with a homogeneous group from normoxia to severe hypoxia. The alveolar gases were confirmed with arterial gases, verifying this

technique at extreme hypoxia. The linear relationships described for PO2 PCO2, and PO2, PCO2 and barometric pressure argue against an increased ventilatory response at severe hypoxia to maintain a minimum PO2 as has been postulated (19). Inspite of marked increase in alveolar ventilation and a very low PCO2 this is insufficient to prevent a further decrease in PO2 at increasing altitude. Nevertheless despite the extreme hypoxia at extreme altitude reported in this study six subjects were able to exercise vigorously at the summit. The arterial pH data indicate that man at extreme altitude is less alkalemic than previously calculated. This study also demonstrated hyperventilation after return to sea level that persists for a prolonged period.

In conclusion, this study demonstrated a linear relation between PCO2 and PO2 and a linear relation between PO2, PCO2 and barometric pressure (for both alveolar and arterial samples) for gradual decompression to extreme altitude in an hypobaric chamber. Thus, it would seem that there must be a critical altitude where the ventilatory response to hypoxia would be inadequate in providing a PO2 necessary to sustain life. This altitude must be higher than a barometric pressure of 240 torr. The fact that our subjects were able to reach 240 torr on several occasions and exercise vigorously for sustained periods supports this conclusion.

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The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy, and or decision, unless so designated by other official documentation.

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LEGENDS

Figure 1 - CORRELATION OF ARTERIAL AND ALVEOLAR SAMPLES. A) Arterial CO2 vs Alveolar CO2. B) Arterial PO2 vs Alveolar PO2. Values plotted represent individual subjects values. RSq=R squared, INT= intercept, and Lin= slope for the linear regression line.

Figure 2 - CORRELATION OF ALVEOLAR AND END TIDAL SAMPLES. A) Alveolar PCO2 vs End Tidal PCO2. 2B - Alveolar PO2 vs End Tidal PO2. Values plotted represent means for all subjects. RSq=R squared, INT= intercept, and Lin= slope for the linear regression line.

Figure 3 -RELATIONSHIP BETWEEN PCO2 AND PO2

A) Sea Level to Pb=240 torr.

B) Below Pb=575 torr. Regression analysis shows a linear relation between Alveolar PO2 and PCO2 and Arterial PO2 and PCO2 (RSq=R squared, INT=intercept and Lin=slope for the linear regression line).

Figure 4 - PO2 AND PCO2 VS BAROMETRIC PRESSURE BELOW 575 TORR. Regression analysis shows a linear relation for PO2 and PCO2 and barometric pressure for arterial and alveolar samples. Arterial PO2, RSq=.986,slope=.11,p<.001, Alveolar PO2, RSq=.934,slope=.109, p<.001, Arterial PCO2, RSq=.980, slope=.0849, P<.001, Alveolar PCO2, RSq=.980, slope=.087, p<.001.

Figure 5 - ALVEOLAR GASES AND ALTITUDE BY DAY AT ALTITUDE.

Altitude in torr x 0.1. There are no significant differences over time at equivalent altitudes.

Figure 6 - ALVEOLAR GASES UPON RETURN TO SEA LEVEL

A)PCO2

B)PO2

The solid line represents values of the six subjects who went to Pb=240 torr. The dashed lines are the values of the subject removed at Pb=282 torr. Alveolar gases on that subject were collected for ten days after return to sea level.

Figure 7 - ALVEOLAR PO2 AND PCO2 FOR THE SEVEN SUBJECTS WHO WENT TO Pb=282 COMPARED WITH SUBJECT 7 WHO COLLAPSED AT Pb=380. Values labeled others represent the mean values for subjects who made it to Pb=282. Values for subject 7 represent his alveolar gases up to the day he collapsed. From this diagram it is seen despite subject 7 being an competitive endurance athlete, his ventilatory response to hypobaric hypoxia was less than the other subjects.

Figure 8 - ALVEOLAR PO2 VS PCO2 FROM PREVIOUS STUDIES AND OEII. Data from Rahn and Otis listed by original source. Close agreement is observed between the various studies with the exception of a few data points from previous studies taken on only one or two individuals.(1-6,8-10,12-19).

TABLE 1

MEAN VALUES OF ALVEOLAR AND ARTERIAL SAMPLES

				ALVEOLAR SAMPLES			ARTERIAL SAMPLES		
DAY	Pb	N	PiO2	PAO2	PACO2	R	PaO2	PaCO2	Ph
1	760	8	149	104.3 ± 9.9	38.2 ± 4.3	.85 ± .13	109 ± 8.3	33.2 ± 2.4	7.42 ± .02
3	575	8	110	63.2 ± 9.0	38.2 ± 3.4	.79 ± .12			
4	501	8	95	53.8 ± 3.8	37.6 ± 3.9	.90 ± .13			
5	482	8	91	53.8 ± 5.3	31.6 ± 2.9	.83 ± .10	58.0 ± 3.9	32.3 ± 2.5	7.44 ± .02
6	463	8	87	50.0 ± 7.1	29.5 ± 3.7	.77 ± .09			
7	447	8	84	46.9 ± 5.0	28.5 ± 2.8	.74 ± .09			
9	428	8	80	47.5 ± 5.4	26.4 ± 3.7	.80 ± .13	52.4 ± 4.0	24.9 ± 2.2	7.46 ± .02
12	412	8	76	48.7 ± 7.7	25.0 ± 4.8	.94 ± .28			
13	397	8	73	41.0 ± 6.7	25.8 ± 4.2	.78 ± .12			
14	380	8	70	40.6 ± 6.0	25.6 ± 3.5	.88 ± .11			
15	380	8	70	43.4 ± 6.2	23.4 ± 3.9	.89 ± .10			
16	380	8	70	43.4 ± 5.2	22.6 ± 3.0	.85 ± .10			
17	364	7	66	40.3 ± 3.6	23.7 ± 2.5	.91 ± .11			
18	349	7	63	36.7 ± 3.8	23.1 ± 3.0	.85 ± .05			
19	349	7	63	39.5 ± 2.2	18.5 ± 1.8	.74 ± .05	41.0 ± 3.3	20.0 ± 2.8	7.50 ± .04
20	349	7	63	35.7 ± 5.4	21.3 ± 3.9	.74 ± .04			
21	380	7	70	45.0 ± 4.6	22.0 ± 3.4	.89 ± .13			
22	349	7	63	36.9 ± 4.0	21.2 ± 3.2	.77 ± .02			
23	349	7	63	35.2 ± 2.9	22.0 ± 2.9	.74 ± .04			
24	347	7	63	38.0 ± 3.6	19.9 ± 2.4	.77 ± .07			
25	335	7	60	35.7 ± 3.2	20.5 ± 2.9	.80 ± .05			
26	314	7	56	31.2 ± 2.9	18.6 ± 2.0	.72 ± .06			
27	306.5	7	54	30.6 ± 3.0	17.5 ± 2.1	.69 ± .04			
28	308	7	55	34.3 ± 3.8	16.4 ± 2.6	.78 ± .08	36.0 ± 4.3	17.3 ± 2.6	7.50 ± .04
30	295	7	52	32.4 ± 4.4	14.9 ± 3.4	.72 ± .05			
31	289	7	51	31.9 ± 3.1	17.0 ± 1.8	.89 ± .03			
32	282	7	49	31.0 ± 3.1	14.4 ± 1.9	.77 ± .09			
33	282	7	49	28.7 ± 2.8	15.3 ± 1.9	.70 ± .04			
34	282	6	49	32.7 ± 3.3	14.0 ± 2.5	.83 ± .03			
40	282	6	49	32.6 ± 3.9	14.2 ± 3.2	.84 ± .05			
35-40	282	6	49	34.9 ± 2.6	14.6 ± 1.2	1.08 ± .21	35.8 ± 1.7	13.3 ± 1.3	7.53 ± .02
34	273	6	47	30.2 ± 2.7	14.7 ± 2.2	.85 ± .06			
34	263	6	45	31.0 ± 3.0	13.9 ± 2.3	1.02 ± .10			
35-40	252	6	44	32.5 ± 1.6	12.2 ± 0.8	1.28 ± .21	32 ± 2.7	10.9 ± 1.5	7.54 ± .02
34	247	6	42	30.8 ± 4.0	12.9 ± 2.4	1.45 ± .59			
35-40	240	6	42	29.8 ± 3.4	12.4 ± 1.9	1.51 ± .89	29.3 ± 2.4	11.7 ± 1.5	7.56 ± .02
40	757	6	148	127.9 ± 5.2	18.1 ± 3.1	.89 ± .14			
41	761	6	149	130.7 ± 5.7	25.5 ± 4.3	1.70 ± .56			
42	776	6	152	132.5 ± 5.7	27.3 ± 4.8	1.59 ± .27			
43	764	5	150	128.9 ± 4.9	27.5 ± 4.6	1.46 ± .20			

Day indicates days at altitude. Pb is barometric pressure in torr.
R is respiratory quotient. Values from day 35-40 represents values from individual ascents.

TABLE 2A

ALVEOLAR GASES UPON RETURN TO SEA LEVEL

	PAO2 (TORR) mean ± s.d.	PACO2 (TORR) mean ± s.d.
PB = 282 TORR	32.56 ± 3.9	14.24 ± 3.2
IMMEDIATE S.L.	127.98 ± 5.2	18.1 ± 3.1
18 HOURS S.L.	130.71 ± 5.7	25.48 ± 4.3
42 HOURS S.L.	132.45 ± 5.7	27.30 ± 4.8
72 HOURS S.L.	128.95 ± 4.9	27.50 ± 4.6

TABLE 2B

ALVEOLAR AND ARTERIAL GASES UPON RETURN TO SEA LEVEL FOR SUBJECT REMOVED AT 282 TORR

TIME	PB	PAO2	PACO2	PaO2	PaCO2	pH	BE	HCO3
	282	28.3	16.7					
4 HOURS	SL			119.6	12.3	7.45	-9.8	12.3
20 HOURS	SL	137.3	23.1	122.5	23.3	7.42	-8.6	14.7
53 HOURS	SL	127.5	26.4	111.8	25	7.43	-6.3	16.6
7 DAYS	SL	124.4	20.02					
8 DAYS	SL	130.8	31.4					
9 DAYS	SL	129.9	32.6					
10 DAYS	SL	120.24	35.5					

A) Alveolar gases for the successful summiters. Samples at Pb 282 were taken immediately before descent.
B) Alveolar and arterial gases for the subject who was removed from the study at Pb 282 torr. BE is base excess.

TABLE 3

INDIVIDUAL ALVEOLAR AND ARTERIAL BLOOD GASES

Pb 760, PIO2 149

SUB	PAO2	PACO2	PaO2	PaCO2	PH
1	110.0	32.3	100.80	37.70	7.39
3	102.2	42.0	110.30	32.40	7.42
4	111.5	32.0	106.00	32.30	7.42
5	111.0	35.6	104.55	33.40	7.42
6	86.3	41.2	110.80	30.10	7.42
7	93.5	41.1	104.30	32.50	7.43
8	105.0	39.5	128.00	31.40	7.43
9	106.2	40.7	107.90	35.60	7.46
MEAN	103.21	38.05	109.08	33.17	7.42
SD	9.02	4.13	8.33	2.42	.02

Pb 482, PIO2 91

SUB	PAO2	PACO2	PaO2	PaCO2	PH
1	54.1	30.0	54.90	35.10	7.46
3	53.2	33.2	60.70	30.30	7.44
4	55.8	27.5	61.70	31.90	7.43
5	63.3	28.1	56.20	30.00	7.44
6	46.6	35.0	60.80	32.40	7.47
7	48.2	33.9	62.70	29.30	7.45
8	57.4	30.5	55.10	36.20	7.43
9	51.3	34.4	52.00	33.40	7.42
MEAN	53.74	31.57	58.01	32.33	7.44
SD	5.31	2.93	3.93	2.47	.02

Pb 428, PIO2 80

SUB	PAO2	PACO2	PaO2	PaCO2	PH
1	57.7	19.8	51.80	23.00	7.47
3	42.5	31.0	47.80	28.70	7.44
4	48.6	21.4	57.00	22.30	7.46
5	48.7	30.4	48.50	27.30	7.44
6	42.8	27.1	53.80	23.90	7.46
7	42.7	27.7	48.90	25.60	7.45
8	44.9	28.7	52.20	25.20	7.46
9	51.8	23.7	58.90	23.70	7.50
MEAN	47.46	26.22	52.36	24.96	7.46
SD	5.37	4.14	4.04	2.19	.02

Pb 349, PIO2 63

SUB	PAO2	PACO2	PaO2	PaCO2	PH
1	46.7	17.3	44.00	15.90	7.57
3	39.4	19.9	38.20	22.50	7.46
4	41.7	16.8	45.50	17.70	7.50
5	38.4	22.5	41.80	22.50	7.48
6	34.8	23.6	42.45	19.50	7.49
8	23.4	24.0	36.40	23.15	7.48
9	38.9	20.5	39.00	18.60	7.52
MEAN	39.0	20.7	41.05	19.98	7.50
SD	4.4	2.9	3.29	2.79	.04

Pb 308, PIO2 55

SUB	PAO2	PACO2	PaO2	PaCO2	PH
1	34.9	17.1	31.40	20.80	7.47
3	28.8	20.9	30.90	20.40	7.49
4	39.3	14.3	37.80	16.60	7.48
5	36.7	13.8	41.40	13.80	7.59
6	32.1	17.4	36.20	17.80	7.49
8	31.0	17.6	33.40	16.90	7.49
9	37.5	13.9	40.80	14.80	7.50
MEAN	34.3	16.4	35.99	17.30	7.50
SD	3.8	2.6	4.27	2.62	.04

Pb 282, PIO2 49

SUB	PAO2	PACO2	PaO2	PaCO2	PH
1	37.8	13.7	35.40	13.22	7.53
3	36.5	14.2	36.30	13.60	7.53
4	37.8	12.6	36.50	12.00	7.55
4	34.4	14.1	34.20	11.80	7.57
6	33.0	15.5	39.60	11.80	7.50
8	31.4	15.5	33.60	14.40	7.53
9	36.7	16.2	35.00	15.20	7.51
9			35.80	14.00	7.53
MEAN	35.4	14.5	35.80	13.25	7.53
SD	2.5	1.3	1.83	1.29	.02

Pb 252, PIO2 44

SUB	PAO2	PACO2	PaO2	PaCO2	PH
1	32.5	12.7	31.20	12.00	7.53
3	32.3	11.7	33.00	10.20	7.52
4	34.7	11.0	36.60	9.20	7.58
6	31.5	11.8	32.60	9.17	7.53
9	30.9	12.2	29.60	12.20	7.53
9	28.9	12.9	29.80	12.40	7.53
MEAN	31.8	12.0	32.13	10.86	7.54
SD	1.9	.7	2.59	1.52	.02

Pb 240, PIO2 43

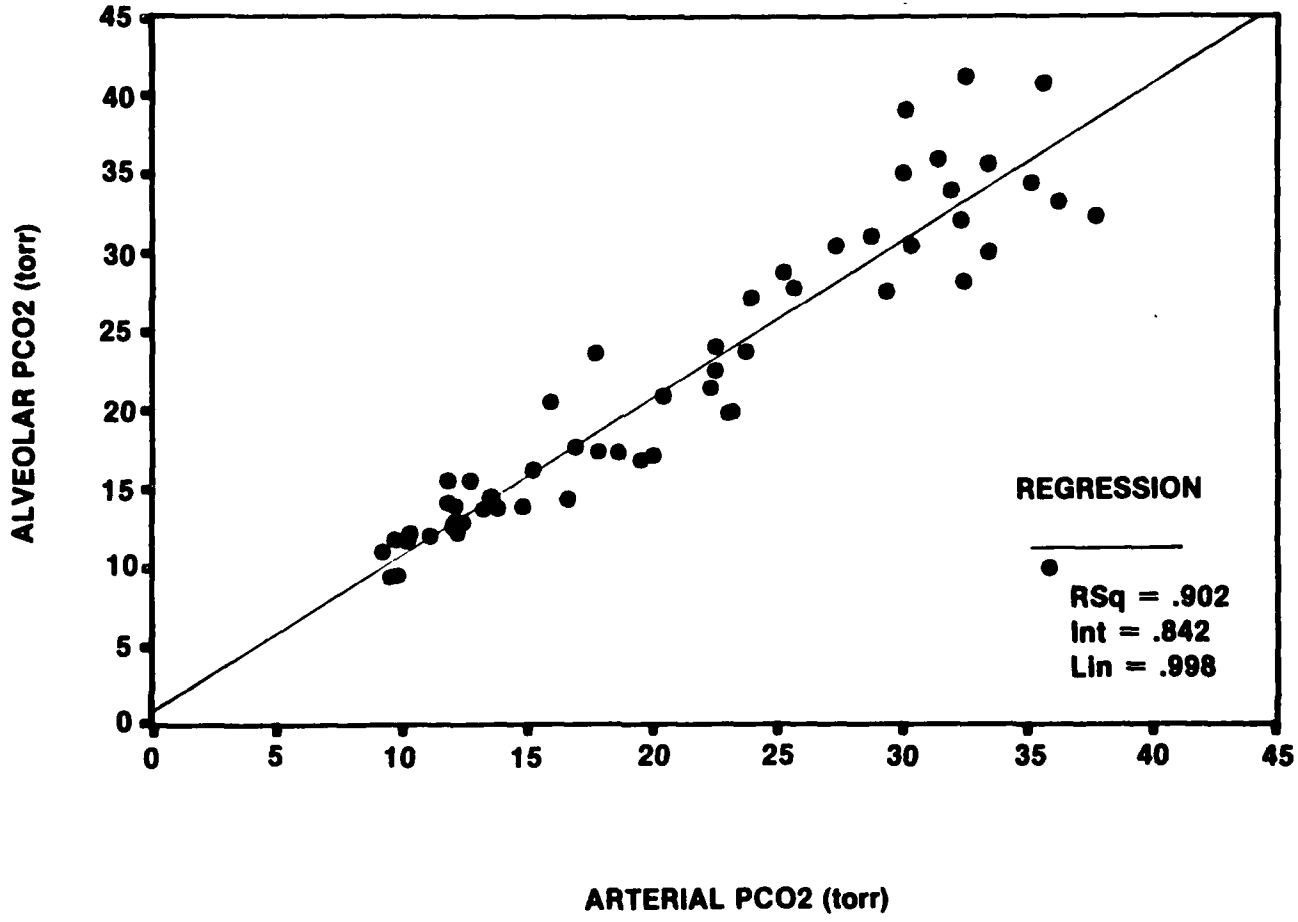
SUB	PAO2	PACO2	PaO2	PaCO2	PH
1	32.6	9.5	31.70	9.80	7.57
1			29.60	11.70	7.51
3	30.7	12.0	29.80	11.10	7.57
3	26.2	13.9	26.10	13.80	7.57
4			26.50	12.30	7.57
4	33.0	9.8	31.50	9.50	7.61
6	28.5	12.1	33.40	10.30	7.54
8	24.4	14.4	28.60	13.60	7.53
9	28.9	12.9	27.30	12.60	7.54
9			28.80	12.10	7.55
MEAN	29.2	12.1	29.33	11.68	7.56
SD	3.2	1.9	2.37	1.50	.02

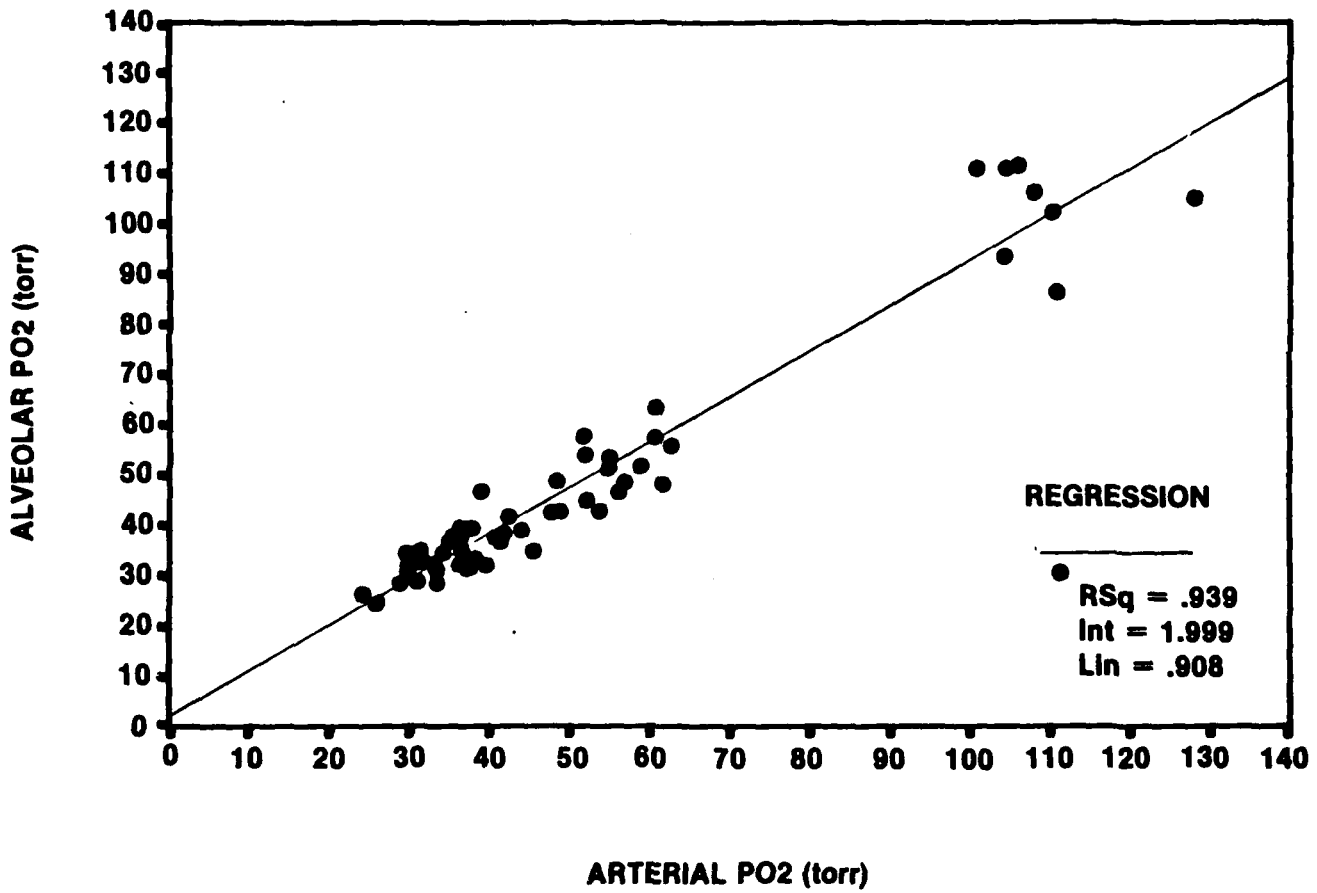
Sub=subject, Pb=barometric pressure in torr, PIO2=inspired oxygen pressure in torr.
Arterial and alveolar samples fro 760-249 torr were not simultaneous.

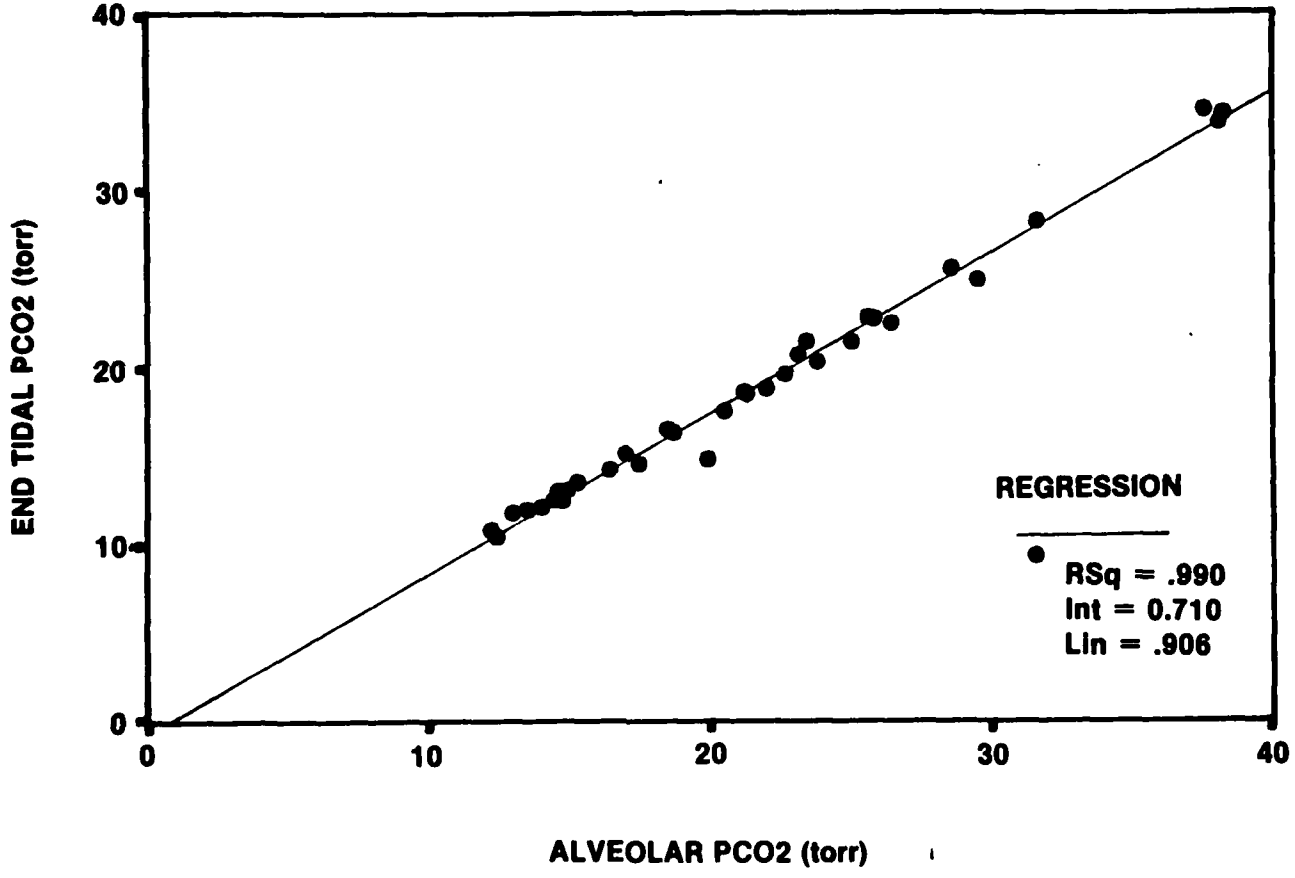
TABLE 4 - Alveolar PO2 and PCO2 in acclimatized subjects from recent studies

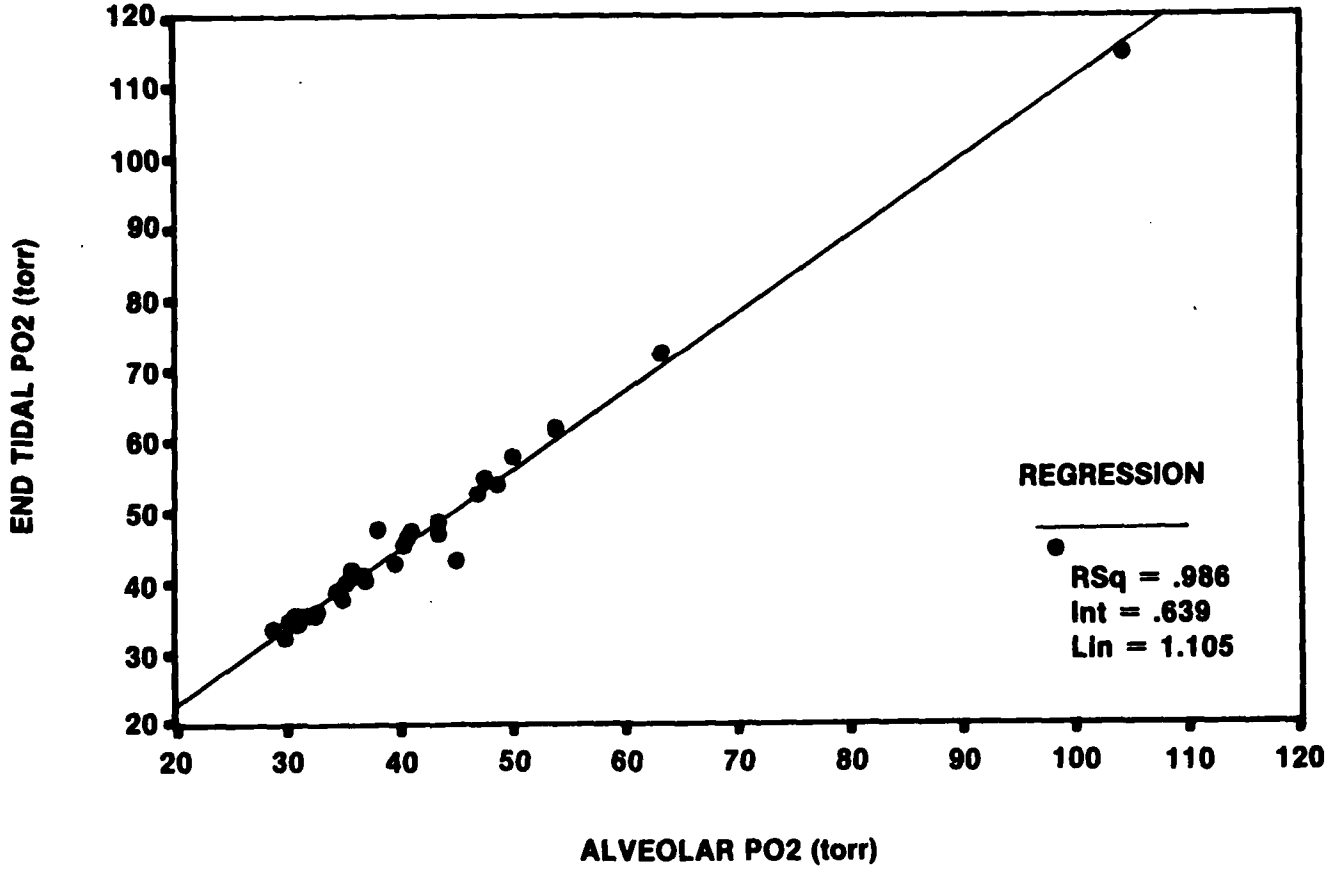
Source	Pb	PO2	PCO2	N
PUGH, 1957	347	39.3	21	
	337	35.5	21.3	
	308	34.1	16.9	
GILL et al, 1962	344	38.1	20.7	4
	300	33.7	15.8	8
	288	32.8	14.3	3
WEST et al, 1981	284	36.1	11	4
	267	36.7	8	1
	253	37.6	7.5	1

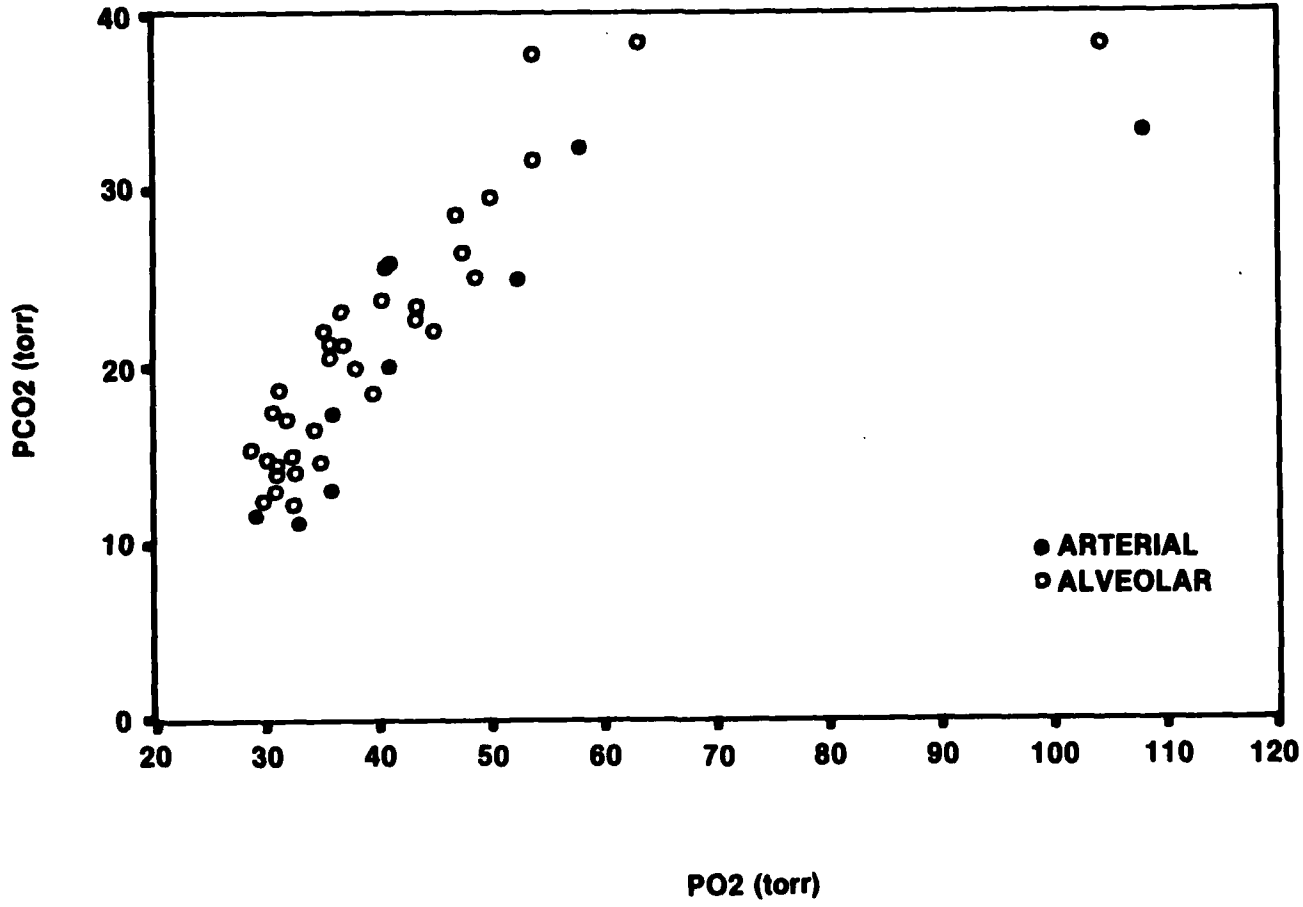
Pb represents barometric pressure, N represents sample size. PO2 and PCO2 represent alveolar values in torr.

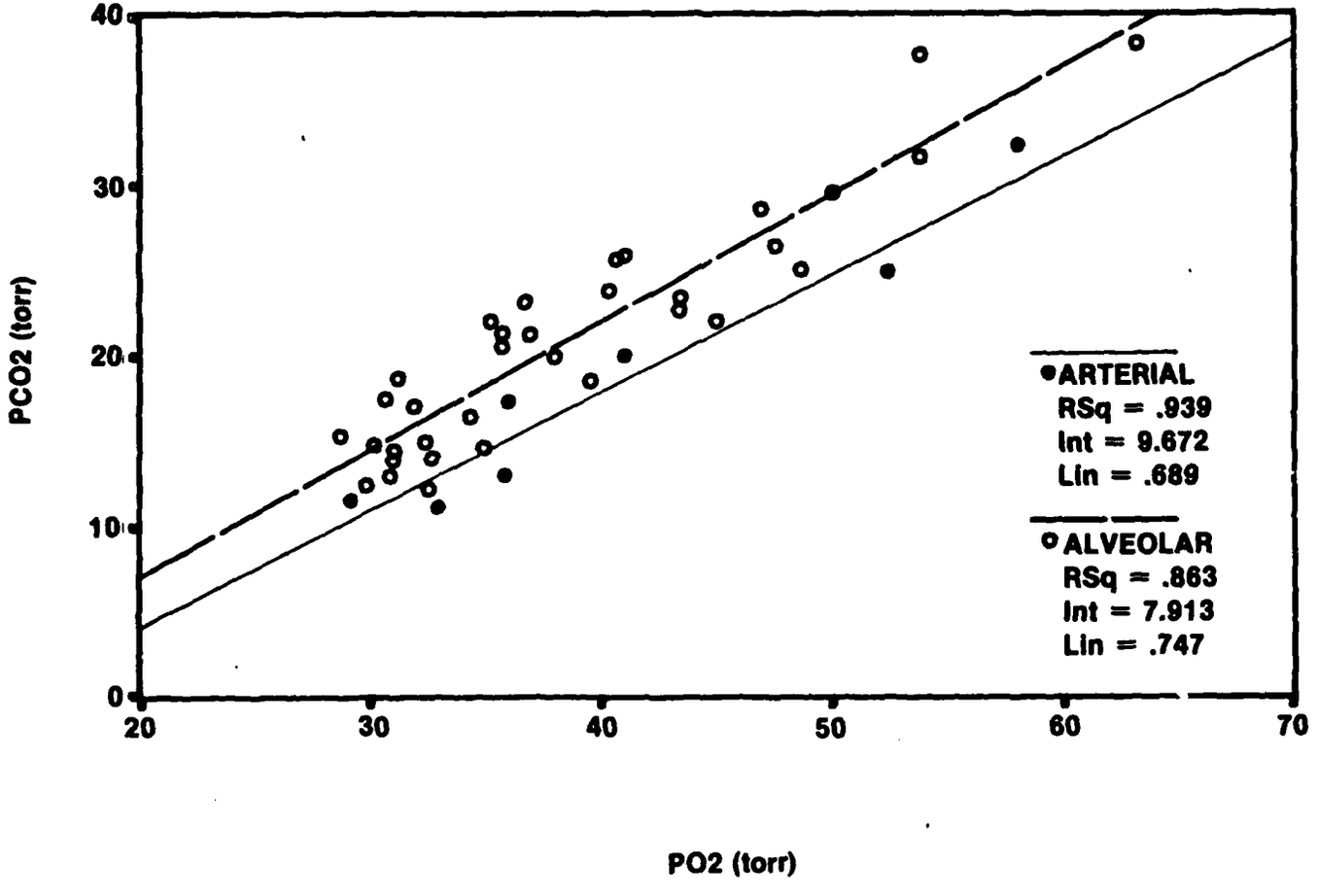

ALVEOLAR PCO2 (torr)
ARTERIAL PCO2 (torr)
0
5
10
15
20
25
30
35
40
45
REGRESSION
RSq = .902
Int = .842
Lin = .998

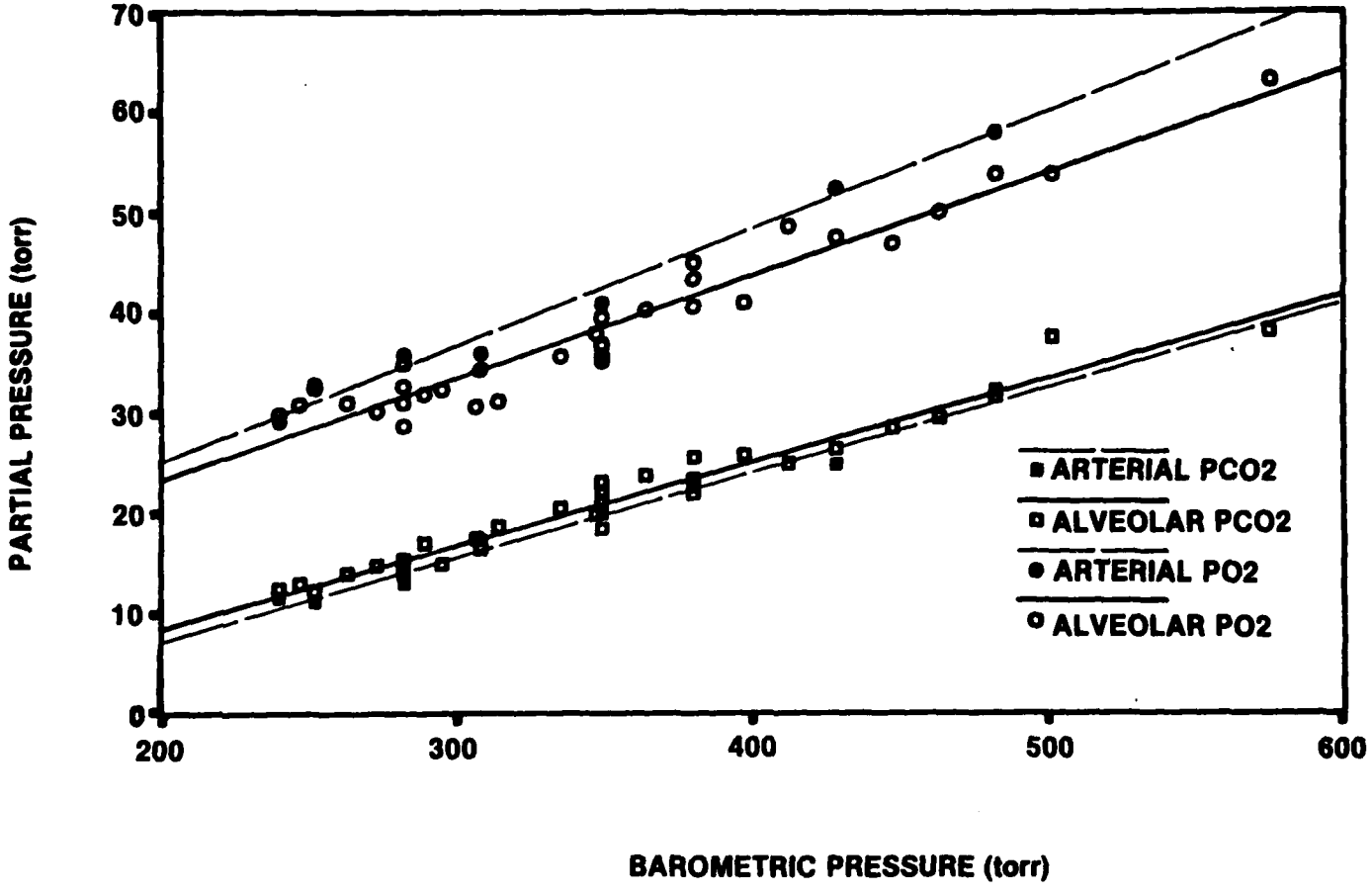

ALVEOLAR PO2 (torr)
ARTERIAL PO2 (torr)
0
10
20
30
40
50
60
70
80
90
100
110
120
130
140
REGRESSION
RSq = .939
Int = 1.999
Lin = .908

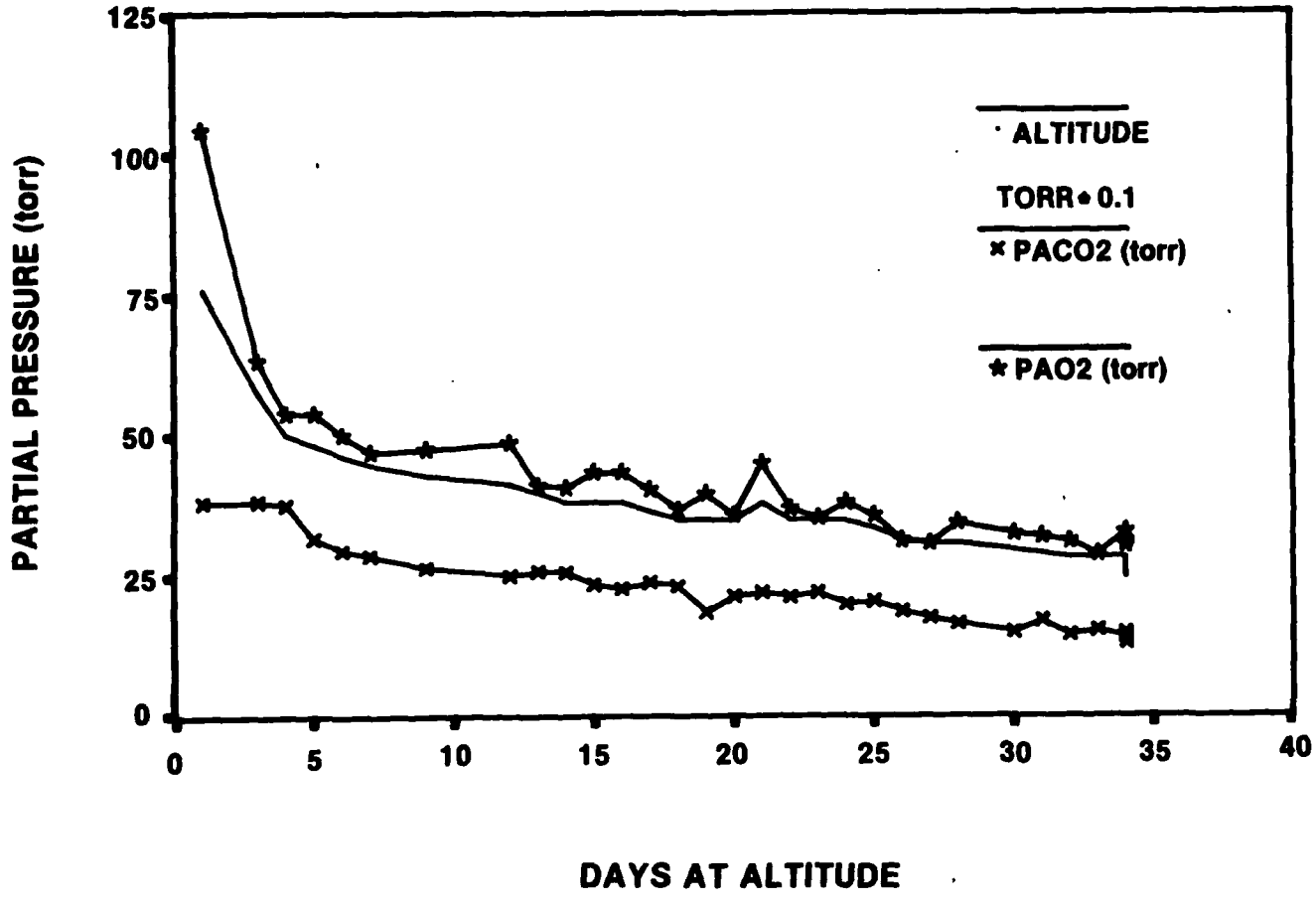

40
30
20
10
0
0
10
20
30
40
END TIDAL PCO2 (torr)
ALVEOLAR PCO2 (torr)
REGRESSION
RSq = .990
Int = 0.710
Lin = .906

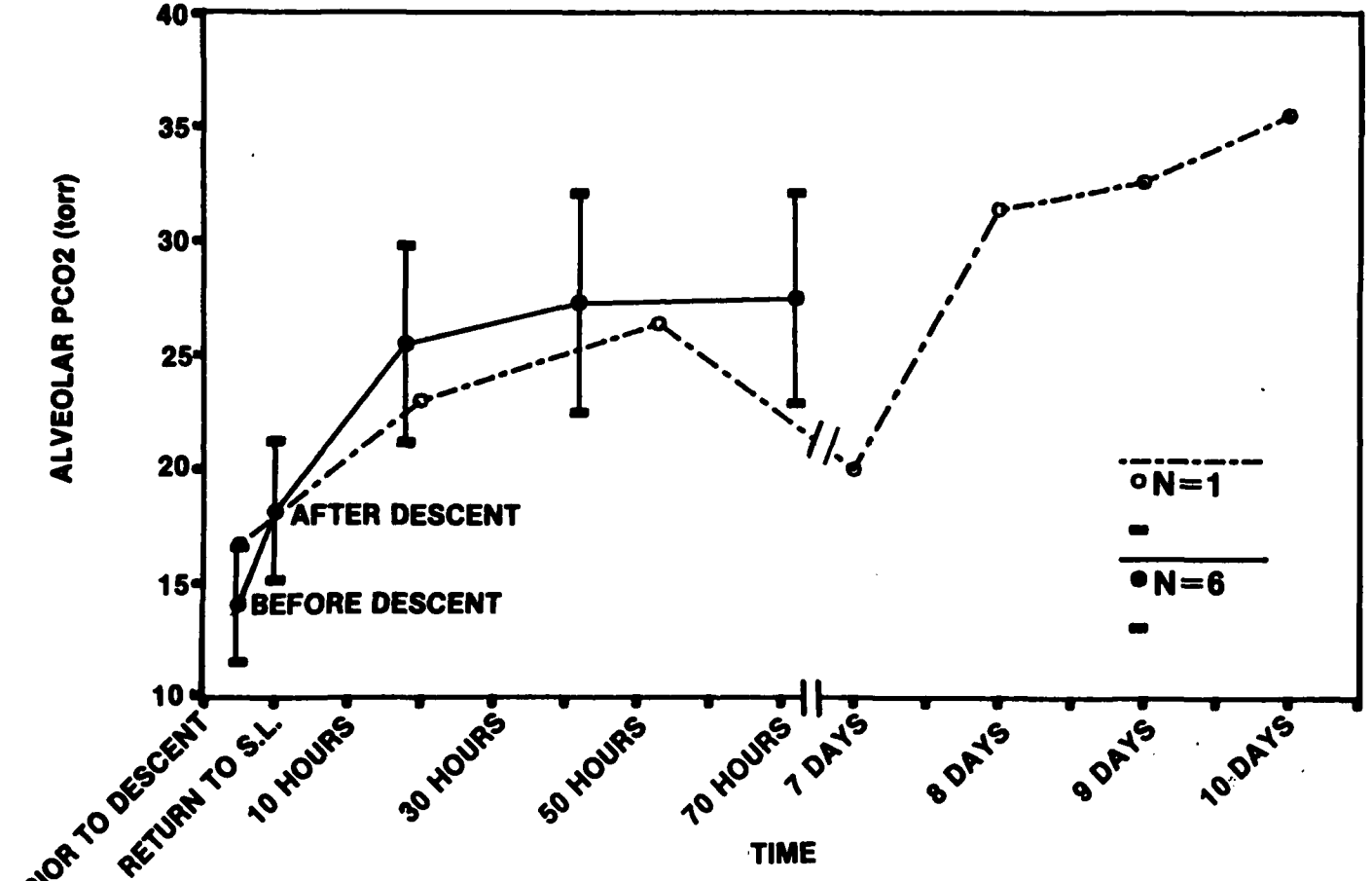

END TIDAL PO2 (torr)
120
110
100
90
80
70
60
50
40
30
20
20
30
40
50
60
70
80
90
100
110
120
ALVEOLAR PO2 (torr)
REGRESSION
RSq = .986
Int = .639
Lin = 1.105

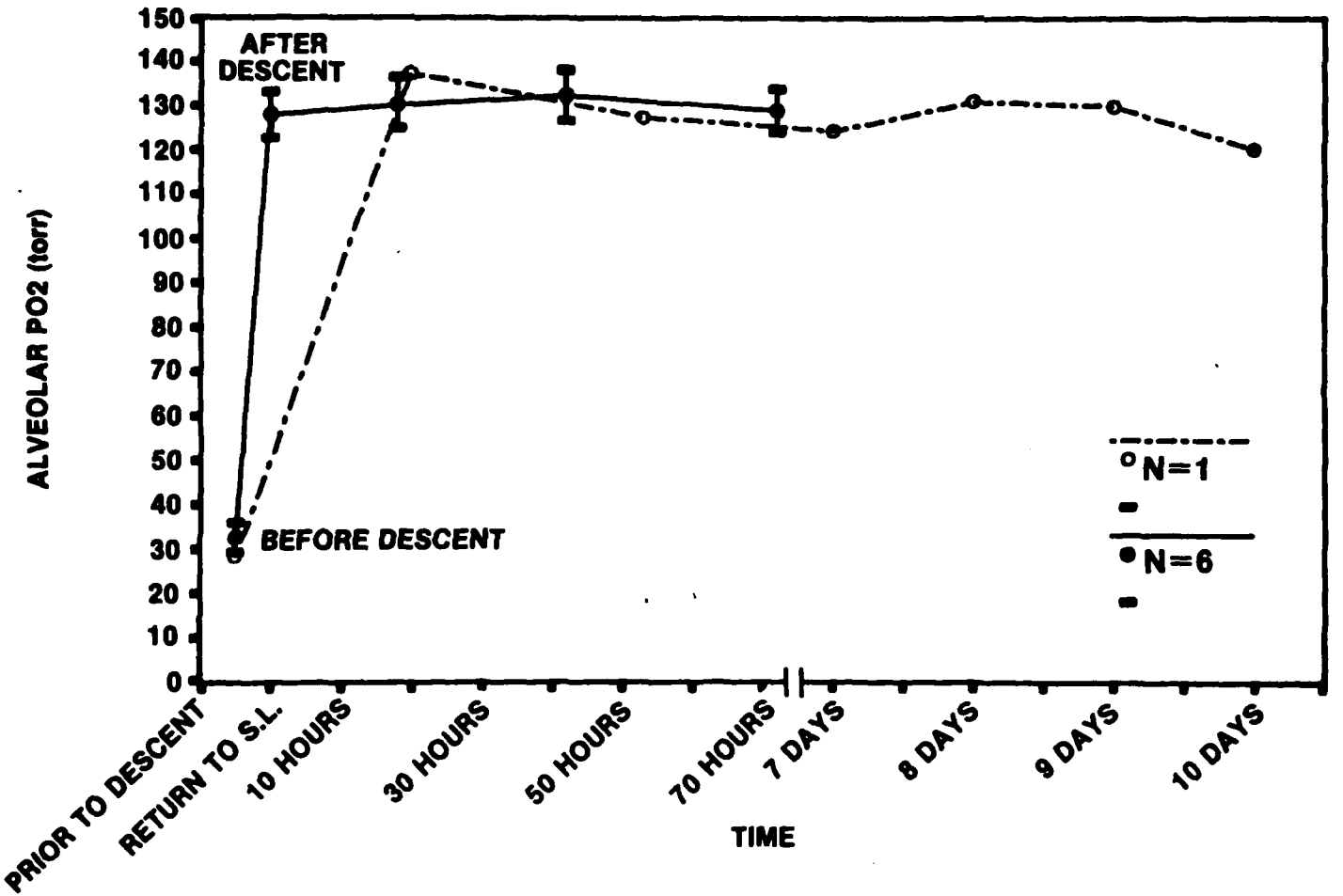

40
30
20
10
0
PCO2 (torr)
20
30
40
50
60
70
80
90
100
110
120
PO2 (torr)
ARTERIAL
ALVEOLAR

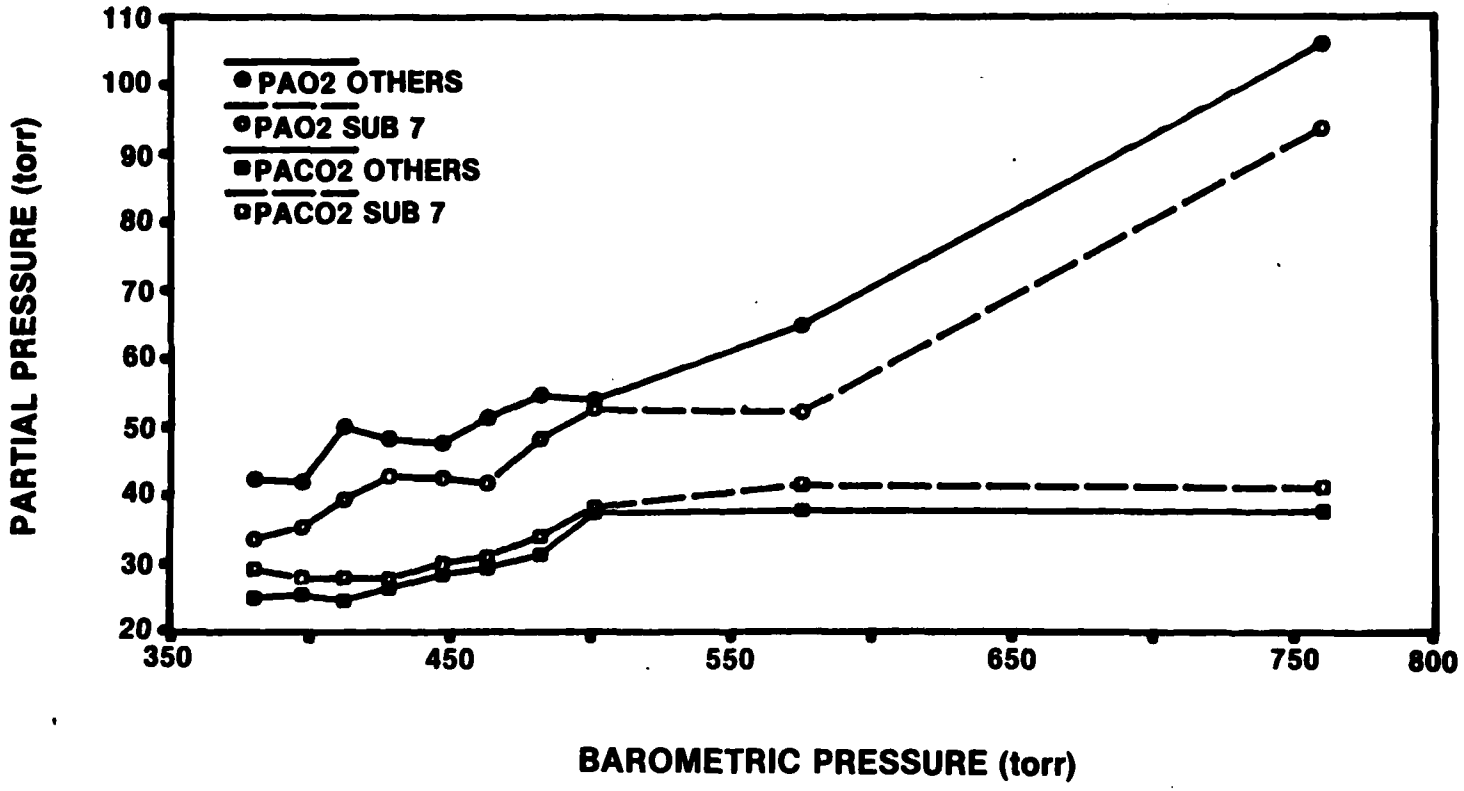

40
30
20
10
0
20
30
40
50
60
70
PCO2 (torr)
PO2 (torr)
ARTERIAL
RSq = .939
Int = 9.672
Lin = .689
ALVEOLAR
RSq = .863
Int = 7.913
Lin = .747

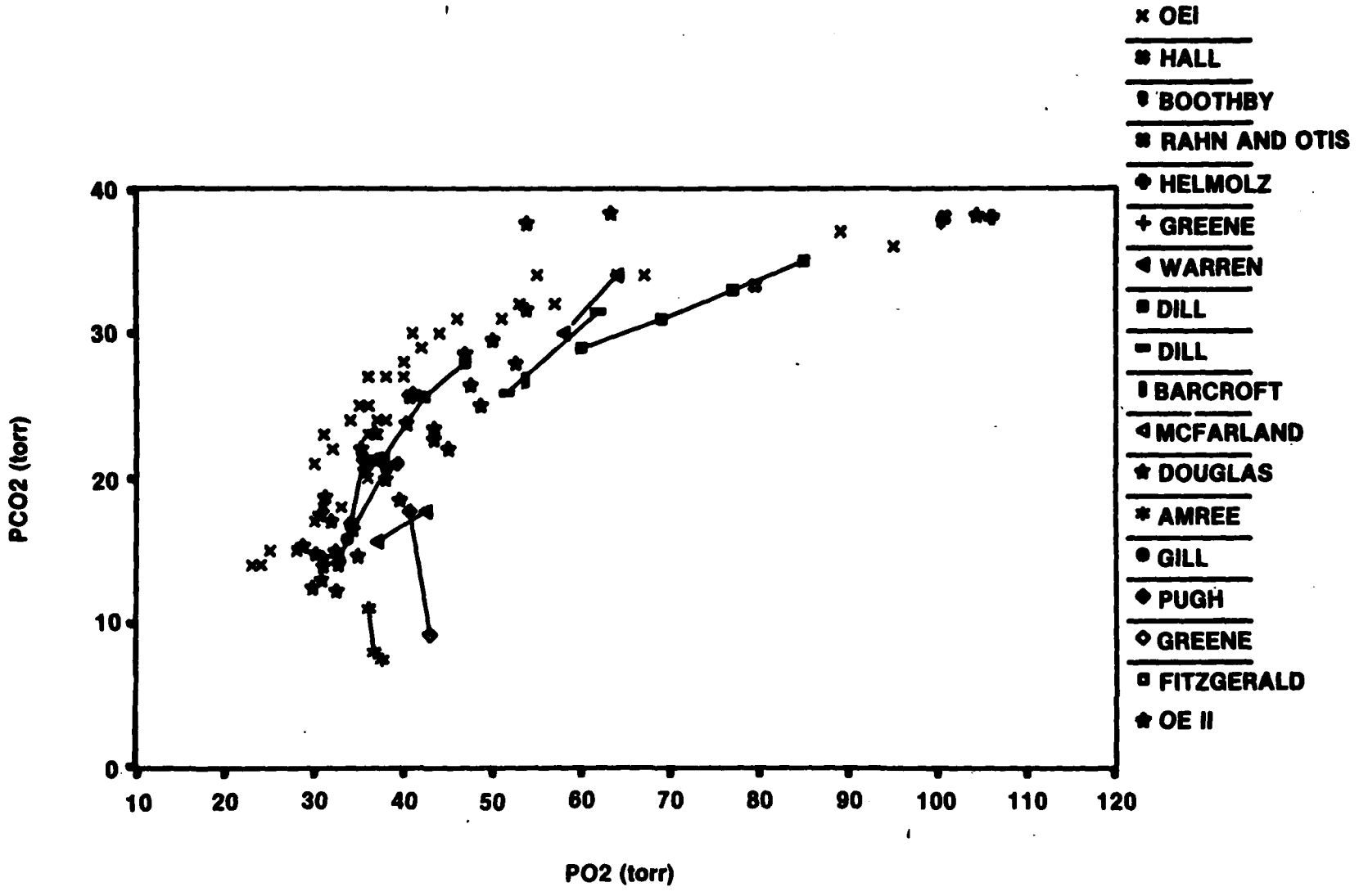

70
60
50
40
30
20
10
0
200
300
400
500
600
PARTIAL PRESSURE (torr)
BAROMETRIC PRESSURE (torr)
ARTERIAL PCO2
ALVEOLAR PCO2
ARTERIAL PO2
ALVEOLAR PO2

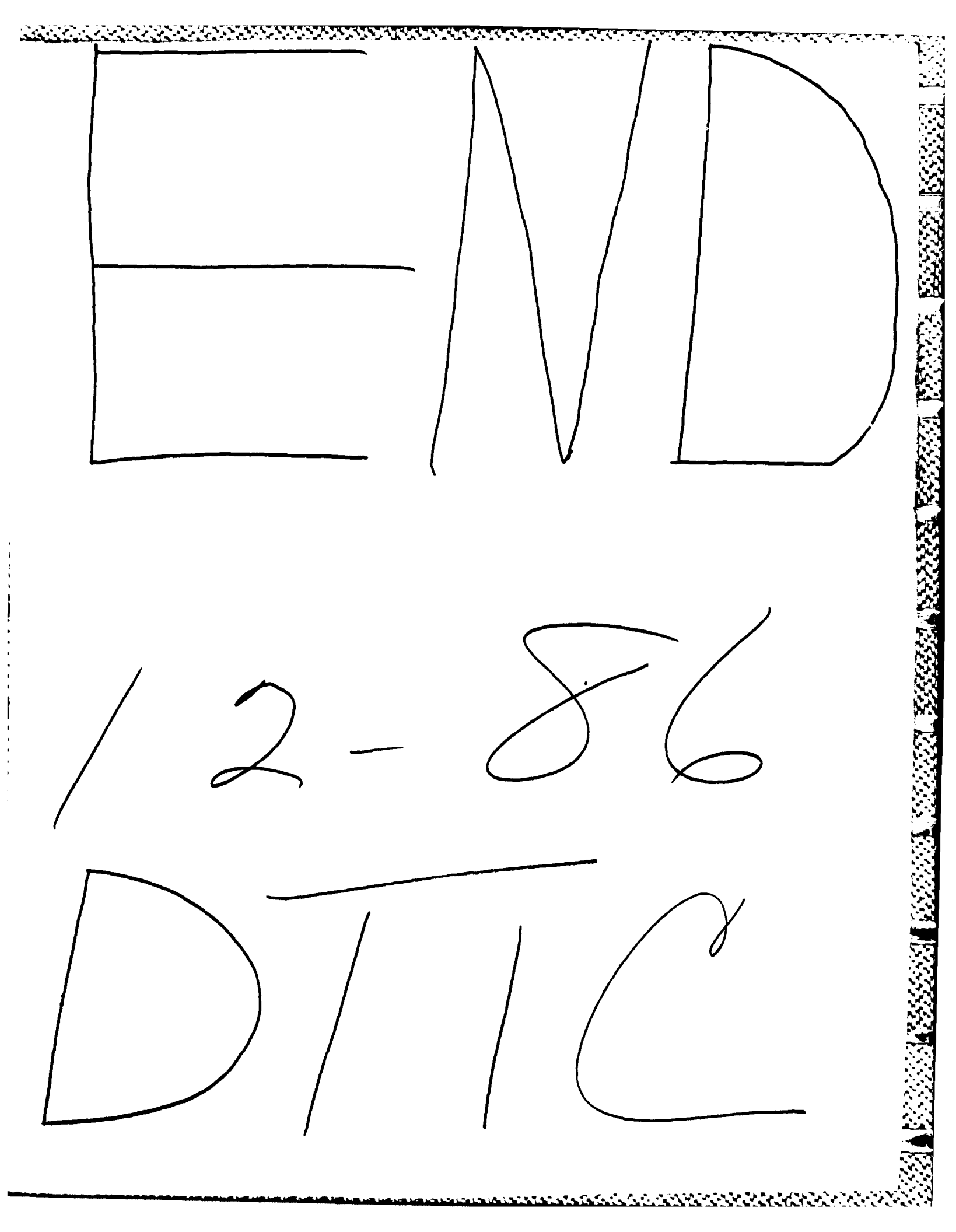

125
100
75
50
25
0
0
5
10
15
20
25
30
35
40
PARTIAL PRESSURE (torr)
DAYS AT ALTITUDE
ALTITUDE
TORR • 0.1
× PACO2 (torr)
* PAO2 (torr)


ALVEOLAR PCO2 (torr)
40
35
30
25
20
15
10
AFTER DESCENT
BEFORE DESCENT
N=1
N=6
PRIOR TO DESCENT
RETURN TO S.L.
10 HOURS
30 HOURS
50 HOURS
70 HOURS
7 DAYS
8 DAYS
9 DAYS
10 DAYS
TIME


ALVEOLAR PO2 (torr)
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
AFTER DESCENT
BEFORE DESCENT
N=1
N=6
PRIOR TO DESCENT
RETURN TO S.L.
10 HOURS
30 HOURS
50 HOURS
70 HOURS
7 DAYS
8 DAYS
9 DAYS
10 DAYS
TIME


PAO2 OTHERS
PAO2 SUB 7
PACO2 OTHERS
PACO2 SUB 7
PARTIAL PRESSURE (torr)
110
100
90
80
70
60
50
40
30
20
350
450
550
650
750
800
BAROMETRIC PRESSURE (torr)


PCO2 (torr)
PO2 (torr)
0
10
20
30
40
10
20
30
40
50
60
70
80
90
100
110
120
OEI
HALL
BOOTHBY
RAHN AND OTIS
HELMOLZ
GREENE
WARREN
DILL
DILL
BARCROFT
MCFARLAND
DOUGLAS
AMREE
GILL
PUGH
GREENE
FITZGERALD
OE II


END
12-86
DTIC